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COMMUNICATIONS

PALSIES OF THE CONJUGATE OCULAR MOVEMENTS*

BY

GORDON HOLMES, C.M.G., M.D.,

LONDON.

IN attempting to determine the site and cause of a lesion which affects a conjugate movement of the eyeballs in any direction, we must keep before us the complex physiological apparatus which regulates the normal movements of the eyes. For these occur not only in response to voluntary impulses, but also as reflex movements excited by peripheral stimuli arising in the retinae, in the ocular muscles themselves, in the labyrinths, in the cervical muscles, and even in response to auditory and cutaneous stimuli. Further, while some of these reflex movements, such as the adjustments of our eyes to changes in the position of our heads and bodies, depend on the activity of sub-cortical centres only, others require the intervention of the forebrain cortex.

Our most natural starting point is the cerebral cortex, which is the organ directly concerned in the voluntary acts. From the earliest days of experimental cerebral physiology the existence of certain areas has been recognized in the forebrain, from the excitation of which associated ocular movements result. The best

*Introductory paper read before the Section of Neurology and Ophthalmology of the Royal Society of Medicine on March 10 and 11, 1921.

known of these cortical centres is that in the second frontal gyrus, which, when stimulated, causes, in animals with binocular vision, conjugate movements of the eyeballs towards the opposite side. This area is generally regarded as a motor centre for the ocular movements, but Sherrington and Leyton have found that the reactions elicited from it differ from those obtained from the precentral motor area, the chief differences being the stronger stimulus required to elicit the movement, the less regular response, the deliberate development of the movement, and the fact that the eyes remain for a time in the position into which they move, and come back slowly. Sherrington and Leyton obtained no purely vertical movements from this frontal area, but others have elicited them either directly, or, as Dr. Risien Russell and Bechterew, after section of the lateral rotators of the eyes, or destruction of the abducens nuclei.

There is unequivocal evidence of the existence of this frontal centre in man too. The most demonstrative evidence is afforded by those cases in which an irritant lesion of this region excites clonic spasms or convulsions; in such cases clonic conjugate movements of the eyes to the opposite side are often the most prominent feature. For instance, one man under my own observation, in whom a meningeal gumma compressed the second frontal convolution, was subject to an enormous number of seizures, all of which commenced with, and some only consisted of, conjugate movements of the eyes towards the opposite side. Weakness of the conjugate deviation of the eyes to one side is not uncommonly due to a lesion of this portion of the brain, but it is usually regarded as a temporary symptom only. During the late war I had the opportunity of observing a very large number of men in whom gunshot wounds had damaged the cortex of the second and third frontal gyri, and my experience was that in every case of recent injury of this region, the patient was either unable to move his eyes fully towards the opposite side, or the movement was less prompt and needed more effort, or that the eyes swung back quickly to their primary central position as though the innervation was not strong enough to maintain their lateral deviation. These disturbances seemed to disappear within a few weeks of the infliction of the wound, when the patient was merely tested by asking him to follow a moving object with his eyes; but other tests showed they were more permanent. When he was asked to turn his eyes promptly to the one side on receiving the order "eyes right" or "eyes left," or when the observer, holding up his hands each at an equal distance to the right and left of his visual axes, asked him to look at once at the fingers which moved on either side, it could be seen that though he turned his eyes promptly and without the slightest effort towards the side of the injury, their movements towards the opposite

side were slower, less complete and accurate, and obviously demanded more effort. Further, if the tests were continued for some time deviation in this direction tired quickly. This paresis of the purely voluntary movements of the eyes, or of movement in response to a stimulus that attracted attention, may be a more or less permanent effect of frontal lesions. I have also found it a valuable test in the localization of tumours in this region of the brain.

The frontal oculomotor centre seems to be concerned only with movements of the eyes which are initiated by volition. All the reflex movements, as those excited from the labyrinth and by purely retinal impressions, as well as those higher reflexes in which conscious visual perceptions are concerned, remain intact when it is destroyed.

I have seen no clinical evidence that the vertical movements of the eyeballs are represented in this frontal area. Its destruction has never, in my experience, influenced the upward and downward movements of the eyes, nor has its irritation produced clonic involuntary movements in these directions, though oblique lateral and upward or downward deviation is not uncommon in epilepsy.

A second cortical centre has been described by Sir David Ferrier in the angular gyrus. On stimulation of its anterior portion in monkeys he obtained upward movement of the eyes, downward movement from its posterior part, and lateral movement from the intermediate area. Bernheimer also obtained movements of the eyes in all directions from this region, but many other experimenters, as Lewandowsky, could elicit only conjugate lateral movement to the opposite side.

As far as I am aware, there is no definite evidence of an oculomotor centre in this position in man ; I never saw a gunshot wound of this region of the brain disturb the ocular movements, and my civil experiences point to the same conclusion. I may cite two cases in which special observations were made. In one man, from whom the late Sir Victor Horsley removed a portion of the right angular gyrus, there was absolutely no limitation of the ocular movements after the operation. In a second case, the removal of a large tumour from the left angular gyrus, producing a destruction so deep that a right homonymous hemianopia resulted, did not influence the ocular movements in any way, We may therefore conclude against the existence of a motor centre for the ocular movements in the parietal lobe. I have, however, repeatedly observed a special disturbance of the ocular movements by gunshot wounds in this region, which must, however, be regarded as a manifestation of apraxia and not as a palsy of movement. In some of the most striking cases, as one which I published, there was apraxia of the limbs too. The chief feature of oculomotor apraxia is failure to obey an order to look in any direction, or to turn the

eyes promptly to any point, although the patient is fully aware of its position in space, and though there is no trace of weakness of movement. This condition of ocular apraxia, which we cannot discuss more fully here, is sometimes associated with disturbances of spatial orientation, but I observed it as an isolated symptom in men with lesions in the region of the left angular gyrus.

Schäfer first, and a large number of physiologists after him, obtained conjugate eye movements from the visual area in the occipital lobe, but it is improbable that this area contains a true motor centre of the same nature as that in the frontal lobe. According to Munk, the movements of the eyes elicited from it depend on the production of perceptions in the visual area by the stimulation, the eyes being then reflexly directed towards the point in the outer world to which the perception is projected.

Paretic deviation of the eyes is occasionally associated with lesions of the occipital lobes that produce hemianopia; the patients then look away from their hemianopic side, but this is not constant, and, in my experience, it rarely persists for long. In gunshot wounds of the visual cortex I never found more than a very transient deviation of the eyes towards the seeing side, and it was as a rule noteworthy only when the patients were in a dull or stuperose condition.

There has been considerable discussion on the nature of this deviation of the eyes associated with hemianopia. According to Bard, Roux, and others, it is secondary to the hemianopia, the loss of retinal afferent impressions producing a relative functional depression of the oculomotor centres in the affected hemisphere. But Déjerine and Roussy, who dispute this explanation, have observed conjugate deviation of the eyes associated with a recent occipital lesion in a woman who was blind from birth, and in whom, therefore, visual impressions can never have exerted any influence on their ocular movements. That the occipital lobes contain centres from which ocular movements may be effected is also made probable by the facts that fibres can be traced from them to the midbrain, and that movements of the eyes can be elicited from the calcarine region after removal of the frontal centres. It seems probable that this efferent occipital system is not concerned in willed and purposive movements of the eyes, but that through it, such movements may be excited by visual impressions that reach the cortical level, as those which bring objects perceived by the peripheral portions of the retinae into central vision, and those by which the eyes follow a moving object. The visual cortex is consequently a higher ocular reflex centre, as well as a perceptive area; and the deviation of the eyes towards the seeing side in hemianopia is due to loss of the visual impressions that under normal conditions excite its reflex activity.

It is, however, disease of the brain-stem that produces the most complete and permanent, and consequently the most obvious, disturbances of the conjugate movements of the eyes. The most common form is limitation or loss of the lateral movements to one side, and as it is easier to discuss this, especially with reference to the anatomical site of the causal lesion, than loss of the vertical movements, we will deal with it first.

Paralysis of the Conjugate Lateral Movements of the Eyes

It is well known that inability to move the eyes to one side is frequently associated with disease in the lower and dorsal portion of the pons Varolii, especially when it extends to the neighbourhood of the abducent nucleus of the same side. A lesion, for instance, in the region of the left abducens nucleus may make the patient unable to move either eye to the left of the middle line, that is to contract the left external rectus and the right internal rectus muscles. But these two muscles are innervated from two different nuclei about 3 cms. apart, and the root fibres proceeding from them nowhere come together in the brain-stem. It is therefore necessary to assume that these two nuclei are connected by association fibres which distribute the exciting impulses to the cells of both. We have such association fibres in the dorsal longitudinal bundles; these, it is well known, contain ascending and descending fibres, many of which are intimately connected with the motor nuclei of the ocular nerves. Some of these fibres come from Deiter's nuclei, others ascend from the ventral columns of the spinal cord, but a large proportion of them spring from the nuclei and scattered cells of the tegmentum of the brain-stem, or take origin in the tectum of the midbrain, chiefly in the anterior quadrigeminal bodies. The latter receive retinal fibres that are probably concerned in the reflex movements and adjustments of the eyes. The dorsal longitudinal bundles may be consequently regarded as associational and reflex optic pathways. The next question which arises is the origin of those fibres that convey to the abducens and oculomotor nuclei, the impulses that excite conjugate lateral movements. According to one hypothesis, which is generally accepted, these fibres spring from the cells of the abducens nucleus and ascend in the dorsal longitudinal bundle of the same or of the opposite side to the midbrain. There are, however, certain facts which controvert this view; in the first place the external rectus may be completely paralysed by a lesion that involves its nucleus though the opposite internal rectus is merely weak, and in the second place cases have been recorded of primary or secondary degeneration of all the cells of the abducens nucleus in which the opposite internal rectus acts normally in lateral movement of the eyes. Gerver, too, has found that disappearance

of all the abducens cells, as a result of the tearing out of one sixth nerve in animals, does not affect the functions of the opposite internal rectus. The second hypothesis that assumes the existence of a supranuclear centre in the neighbourhood of the nucleus of the sixth nerve is consequently more probably correct. There is now considerable evidence that this supranuclear centre lies oral and slightly ventral to the abducens nucleus. Into it come all the impulses that can excite conjugate movements of the eyes to the same side; voluntary impulses from the opposite frontal centre, impulses from the occipital and temporal lobes which effect reflex adjustments of the eyes to visual and auditory stimuli, others from the lower visual and auditory reflex systems in the roof of the midbrain, impressions of vestibular origin from Deiter's nucleus, and probably proprioceptive afferent impressions from the neck muscles. All impulses that can excite conjugate lateral movements of the eyes reach this centre, in it they are probably correlated and coordinated to produce an adequate response, and by efferent fibres from it the resultant is transmitted to the motor cells by which the corresponding internal and external recti muscles are innervated. It is probably through it too that the antagonists of the contracting muscles are reciprocally inhibited.

Destruction of this supranuclear mechanism consequently abolishes all voluntary and reflex lateral movements of the eyes to the same side. But these muscles are paralysed for conjugate lateral movement only; we can at least say this of the internal rectus, which still acts normally in convergence of the eyes, though it may fail to contract when lateral movement is attempted. It is less certain in the case of the external rectus, since we have a very limited power of conjugate divergence of the eyes, and since a lesion which destroys the supranuclear centre is liable to injure at the same time its adjacent nucleus. Therefore this supranuclear mechanism has the physiological property common to all motor mechanisms above the level of the final common paths in that it is concerned in the production of movements and not in exciting the contraction of individual muscles.

The various fibres of different function which converge in this supranuclear centre may be injured separately. Lesions of the lateral portion of the pons, for instance, may abolish the reflex turning of the eyes to labyrinthine impressions though the eyes may move normally to volitional and other impulses. But the most striking form of dissociation of lateral conjugate movement is that in which the voluntary deviation of the eyes is lost, but their reflex movements persist. This is presumably due to interruption of the cortico-bulbar fibres above the supranuclear centre, but the exact course that these take is not yet known. In this condition the patient may be unable to move his eyes to one side on command,

though he can follow a slowly moving object in this direction, or, as in one of my cases, his eyes may remain on a stationary object which he fixes when his head is passively turned in the other direction; or his eyes may suddenly swing round in response to a sudden retinal or auditory stimulus. This isolated disturbance of voluntary lateral movement may be produced by cortical or subcortical lesions that destroy the cerebral ocular centres or their efferent fibres, but it is usually then incomplete and transient; here, as is the case with the other cranial motor nerves, the paralysis is greater the nearer the injury of the upper motor neurons is to their nuclei.

If the opposite internal rectus receives its innervation for lateral conjugate movements through fibres which ascend the brain-stem from the region of the sixth nucleus, interruption of these connecting fibres should produce an isolated palsy of the internal rectus in lateral movement, though this muscle may still contract on convergence. I have seen such a paralysis of the internal rectus in conjugate lateral deviation only in two cases; in both it contracted normally in convergence, and in both the symptoms made it probable that the dorsal longitudinal bundles were interrupted in front of the sixth nucleus, but the sites of the lesions could not be confirmed by autopsy.

Paralysis of the Vertical Conjugate Movements of the Eyes

Disturbances of the conjugate vertical movements of the eyes may be treated more briefly, as the physiological mechanisms which control them must be very similar to those of the lateral movements. It is not uncommon for neurologists at least to see loss or limitation, equal in degree in the two eyes, of either upward or downward rotation, or of both, with the lateral movements intact. Upward movements are most frequently affected, and next in order come paralysis of downward movement and of convergence, but all vertical movements of the eyes, with or without disturbance of convergence, are commonly abolished.

Such disturbances of the conjugate vertical movements have been explained by local lesions of the oculomotor nuclei, in which it is usually assumed that the separate ocular muscles are represented by separate groups of cells. Though this may be true, the divergent conclusions arrived at by the numerous authors who have attempted to identify muscular representation in these nuclei, as Hensen and Völker, Perlia, Bernheimer, Bach, Tsuchida and more recently Brouwer, show that we have no definite facts on which to rely. And it is scarcely conceivable that, as happens in the conjugate palsies of the vertical movements, a nuclear lesion could affect only the elevators or the depressors of the eyes, paralyse these to exactly the same degree on the two sides, and not affect the functions of the other muscles innervated by these nuclei. This explanation

becomes even more improbable when we remember that upward movement requires not only the coordinated cooperation of the two elevators of each eye, the two superior recti and the two inferior obliques, but also a parallel elevation of the lids by the levatores palpebrarum; and that the depressors, the inferior recti and the superior obliques, are innervated by two distinct nuclei separated by a relatively considerable space.

We are consequently, in my opinion, forced to the conclusion that, as in the case of the lateral conjugate rotators of the eyes, there are supranuclear mechanisms for upward and downward movements and for convergence, mechanisms which would in fact correspond to the associational cells intercalated between the spinal afferent reflex fibres and the descending spinal tracts on the one hand, and the motor cells of the ventral horns of the cord on the other.

But we are ignorant of the anatomical position of these supranuclear centres. According to one hypothesis they lie in the anterior quadrigeminal bodies; this is based partly on the statements of Adamük, Ferrier and others who elicited conjugate vertical movements by stimulation of this region, or observed loss of them after its destruction, and on the unquestionable fact that the vertical movements of the eyes are frequently disturbed by lesions of the tectum of the mid-brain. But in the great majority of the cases recorded the quadrigeminal lesions were tumours, which may affect indirectly by pressure either the third nuclei or centres in the tegmentum. It is in the tegmentum, or in the central grey matter around the aqueduct of Sylvius, that others would place the supranuclear centre. Freund, for instance, attributed loss of upward movement of the eyes to destruction of Darkschewitsch's nucleus, and the consequent degeneration of the lateral fibres of the dorsal longitudinal bundle, which was present in his case. Even after collating my own experiences with the literature on the subject, I am unable to come to a definite conclusion as to the position of these supranuclear mechanisms for the vertical movements, though I incline to the opinion that they are situated in the anterior quadrigeminal bodies. This opinion is based, in the first place, on the frequency of disturbance of the conjugate vertical movements of the eyes by tumours growing from the pineal body or from the splenium of the corpus callosum, which readily compress the anterior colliculi, and by the observation during the war of three cases of injury of the tectum alone by small penetrating missiles in which the vertical movements of the eyes were affected.

But, whatever may be the anatomical situation of these supranuclear mechanisms, we have considerable evidence of their relative positions, since tumours and other lesions extending from before backwards into the midbrain affect first upward movements of the eyes, then downward movements and finally convergence.

Consequently, we can assume that the centre for elevation lies the most anteriorly, and that for depression the furthest posteriorly. It is interesting in this connection that loss of upward deviation is usually associated with disturbances of the pupillary reactions to light, and that of downward movement frequently with loss of convergence and accommodation. There may be, however, an isolated loss of convergence and accommodation, as in two cases I have seen, and in those recorded by Eales, Parinaud, Stölting and Bruns.

The palsies of the vertical movements may be also dissociated; the most common form is loss of the voluntary movements with reflex movement unaffected. For instance, the patient may be unable to raise or lower his eyes from the horizontal level on command, or by a deliberate effort, but he may do so when a light is suddenly flashed above or below his visual axes, or his eyes may deviate upwards when he closes his lids, or these movements may be elicited by either asking him to follow a slowly moving object, or by bending his head backwards or forwards while his gaze is directed on a stationary object. These involuntary ocular movements evoked by passive movement of the head may represent labyrinthine reactions, but as Schüster found in one of his cases that they occurred only when the head was moved on the vertebral column and not when the head and body were displaced together, they may obviously be reflex adjustments excited by the proprioceptive afferents from the cervical muscles, similar to those which have been investigated by de Kleijn and Magnus. In these dissociated palsies we must assume the interruption in the neighbourhood of the supranuclear centre, of the cortico-bulbar fibres that terminate within it.

We are therefore led to the conclusion that there exists in the upper end of the midbrain, at a higher physiological level than the nuclei of the ocular nerves, a more complex associational or coordinating mechanism which receives all the impulses that can effect vertical and convergent conjugate movements of the eyes, combines and correlates them into an appropriate resultant, and distributes this to the nuclei of those muscles that should contract or relax.

Considerable attention has been directed to disturbances of the ocular movements in cerebellar disease, but they are relatively unimportant, even as clinical signs.

For some days after an acute unilateral injury of the cerebellum, the eyes, while at rest, are generally deviated towards the opposite side, especially if the patient happens to be unconscious, and it is often difficult to make him move them conjugately towards the injured side.

When he attempts this the range of movement is occasionally

incomplete, but usually a more striking feature is its slowness and the effort necessary to execute it. This conjugate paresis may be associated with erroneous projection towards the homolateral side. It diminishes gradually, but the difficulty in movement to this side, in comparison with deviation in the opposite direction, frequently persists for weeks. The vertical movements of the eyes and convergence are never similarly affected.

Occasionally the position known as "skew-deviation" is observed in patients with acute cerebellar lesions, that is the homolateral eye is directed downwards and inwards and the other upwards and outwards. This lack of parallelism in the optic axes disappears on fixation, when this can be obtained, and consequently diplopia does not result. It is produced only by extensive destruction of the cerebellum, and has not, in my opinion, any localizing value.

OCULAR PALSIES*

BY

LESLIE PATON

LONDON.

THE subject of ocular palsies is one of great complexity, and in its consideration the relationship of the oculomotor mechanism to many different parts of the central nervous system has to be taken into account. If we take only one disease, syphilis, and consider the different ways in which it can produce an ocular palsy, it will enable us to realize dimly the complexity of the subject. Syphilis may affect directly any part of the oculomotor apparatus, cortical, supra-nuclear, nuclear, nerve roots, nerve trunks in their intra-cranial course, in the cavernous sinus, or in the orbit, or it may affect the muscles themselves. A gumma may develop at any point. A syphilitic meningitis may affect the nerves. Syphilis sets up disease of the vessels and we may get haemorrhages affecting the nuclei or nerve roots, or a syphilitic arteritis in the internal carotid may press on nerve trunks in the wall of the cavernous sinus, or an endarteritis may affect the vessels supplying the crura, the mid brain or the pons, and give rise to areas of softening; or syphilis may give rise to tabes or general paralysis, and to all the variety of ocular disturbances arising in these diseases. Yet it is doubtful if more than 40 per cent. of cases of ophthalmoplegia are due to syphilis, directly or indirectly.

*Introductory paper read before the Sections of Neurology and Ophthalmology of the Royal Society of Medicine on March 10 and 11, 1921.

Classification of Oculomotor Paralyses

Ocular palsies may be classified in three main groups:—

I. Paralyses of central or cerebral origin, comprising three sub-groups:—

(1) Supranuclear lesions.

(a) Cortical.

(b) Subcortical.

(2) Internuclear lesions.

(3) Nuclear and nerve root lesions.

II. Paralyses of extra-cerebral origin, *i.e.*, of nerve trunks, again comprising two sub-groups:—

(1) Intracranial.

(2) Intraorbital.

III. Paralyses of muscular origin.

It is important in this connection to remember that disturbances of ocular movements may result from lesions affecting the afferent nerves as well as from those affecting efferent nerves, and also, that there are important afferent nerve fibres concerned in the control of ocular movements in the oculomotor nerves, in the optic nerves, in the vestibular nerves, and in the nerves from the muscles of the head and neck.[1]

It is to the sub-group (3) in Group I—Paralyses of nuclear and nerve root origin—that Mauthner applies the term "Ophthalmoplegia" in its more limited sense, and it is more especially with this and the internuclear group that I deal in this paper.

PART I.—ANATOMICAL

The nuclei of the oculomotor nerves lie in the grey matter in the floor of the aqueduct of Sylvius, reaching forwards into the floor of the third ventricle and backwards into the floor of the fourth ventricle. The nucleus of the third nerve passes without a definite interval into the nucleus of the fourth, but the sixth nucleus is distinctly separated from the fourth, and lies in the grey matter in the floor of the fourth ventricle, separated from the ependyma by the curving fibres of the seventh nerve root.

The dorsal or posterior longitudinal bundle coming up from below forms connections with all these nuclei. It runs ventral to and to the inner side of the sixth nerve and ventral to and slightly to the outer side of the fourth and third, and then continues on round the red nucleus. It contains both ascending and descending fibres.

Dorsal to the grey matter in this neighbourhood lies another longitudinal bundle of fibres, the bundle of Schütz.

The third nucleus is composed of two main lateral groups of large cells (about 40 μ. in diameter) forming crescents with the concavity outwards, rounded dorsally and fining off ventrally.

Between the two anterior horns of these crescents, and lying in the more dorsal area, are two groups of smaller cells, almost touching in the middle line. Like the lateral groups, they are broader dorsally and thinner ventrally, and comma shaped antero-posteriorly: these are known as "Edinger-Westphal nuclei." Ventral, and slightly posterior to those, lying exactly in the middle line, is a single spindle-shaped group of large cells—the nucleus of Perlia. A few scattered lateral cells lie outside and amongst the fibres of the posterior longitudinal bundle.

Lying above and to the outer side of the anterior end of the lateral mass, there is another group of cells to which an oculomotor function has been ascribed by Knies[2] — the nucleus of Darkschewitsch.

Numerous commissural fibres pass from the cell groups on the two sides of the mid line, but as we reach the region of the fourth nucleus, these disappear. Though there is no definite separation between the third and the fourth nuclei, there is a space where cells are less numerous, and the region of the fourth is indicated by the disappearance of the crossing fibres.

The researches of Bernheimer[3], Westphal and others seem to indicate that the small-celled mesial nuclei of Edinger and Westphal are concerned in the innervation of the sphincter pupillae, and that the large-celled single median nucleus supplies the ciliary muscles. As the description in most text-books is based on Bernheimer's work, it has come to be accepted in a more dogmatic fashion than is desirable, and it has been strenuously combated by Bach[4], Marina and others. Bach found no signs of degenerative changes in the Edinger-Westphal nucleus after exenteration of the orbit in apes and exenteration of the eye in cats and dogs, and Marina's work on the same lines was confirmatory of Bach's. Tsuchida[5], working under von Monakow, came to the conclusion that there was no definite evidence to show that this nucleus was connected with oculomotor innervation. Brouwer[6] considers that too much stress has been laid on the absence of the obvious degenerative changes in the cells of this nucleus in cases of Argyll Robertson pupil, and in other cases where an ophthalmoplegia interna has co-existed with an ophthalmoplegia externa. He agrees with Westphal and Bernheimer in associating it with the innervation of the iris and adopts the name suggested by Jacobsohn[7]—"Nucleus sympathicus nervi oculo motorii."

Magitot[8], in a recent book on the Iris (Paris, 1921), states that, in his opinion, none of these groups of cells in the third nerve nucleus are concerned in the innervation of the sphincter, since they are not yet developed in the human embryo at a time when the light reflex already exists. Tsuchida has shown that all the accessory nuclei of the third, Edinger-Westphal, Darkschewitsch,

Bechterew, etc., only make their appearance in the seventh month of intra-uterine life, and Magitot has seen the light reflex manifest in premature infants at the beginning of the sixth month.

In Magitot's opinion, the control of the sphincter is by a continuation up into the bulb of the same column of cells which gives rise to the dilator fibres in the cord, the intermedio-lateral tract, and that the whole control of the iris is a function of the sympathetic system, and so can be brought into line with Gaskell's theories.

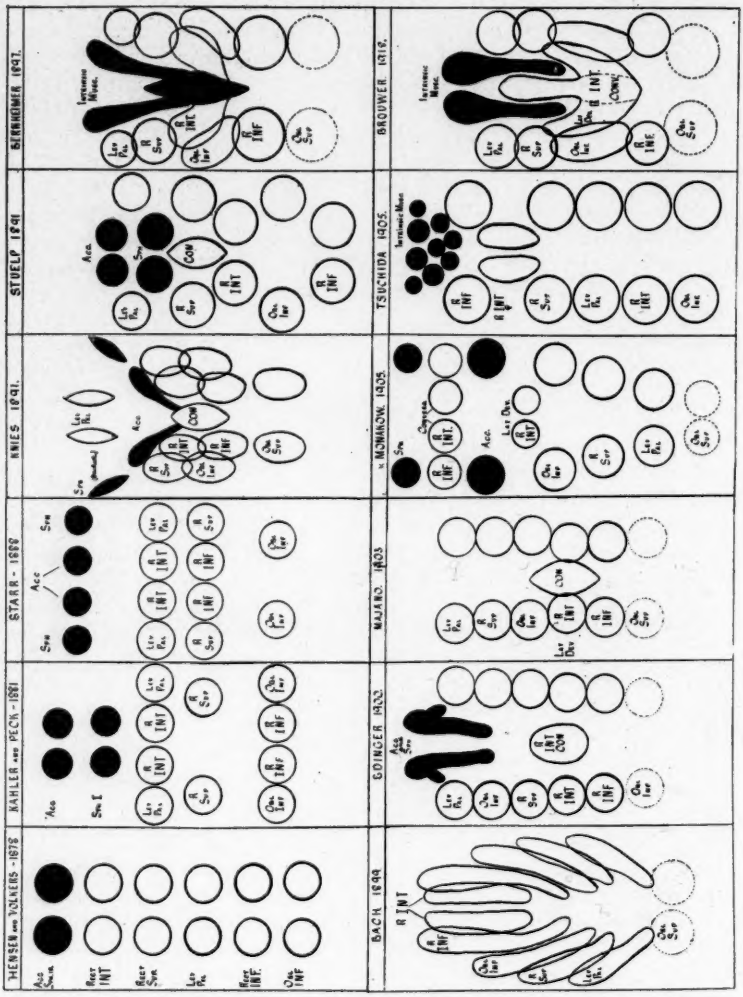
The literature on this subject is enormous, but from the little I have read of it I can only give the Scots verdict of "Not Proven."

With regard to the group of cells described by Darkschewitsch, a more definite assertion can be made that they are not connected with the oculomotor nuclei, but are definitely connected with the fibres which compose the posterior commissure at the back of the third ventricle. According to Bernheimer,[3] they link up the posterior longitudinal bundle with the posterior commissural fibres.

There is no anatomical separation of the large-celled lateral group, and the evidence as to the place of origin of the nerve fibres of the different muscles in different parts is very contradictory. I have copied, for your edification, a diagram from Brouwer's[6] paper already referred to, and it serves to show how various are the views of different workers.

If only on *a priori* grounds, we must assume that the internal recti nuclei lie in that part of the lateral group where the crossing fibres are numerous, and similarly, the inferior oblique and inferior rectus nuclei probably lie where there are crossing fibres. Now, these are most numerous in the posterior half. That would then indicate that the anterior ends of the lateral crescents of large cells are concerned in the innervation of the levator palpebrae superioris and the superior rectus. Then when the middle parts of the crescents come closer and into relationship with the small-celled paired nuclei, and the large median nucleus, the rectus internus is represented, and behind that, where the crossing fibres are numerous, the inferior rectus and the inferior oblique.

When, however, we think how intimately every ocular muscle is concerned in every movement of the eye, and how there is never a movement which does not imply some contraction of one set and some relaxation of another set, we may be forced to the conclusion that each muscle must be more or less represented all through the oculomotor nucleus, and that at least a number of the cells present, and especially the cells of the central nucleus, are in the nature of intercalary cells, linking up the movements of different muscles, and the movements of the two eyes. Now if there are, as I think, intercalary cells present, they would belong to a higher level neuron, and consequently would not suffer degenerative



From Brouwer *Zeitschrift fur die ges. Neur. und Psych.* Vol. XL, 1918.

changes so early as the cells connected with the direct motor neuron, when the nerve fibre originating from it is affected.

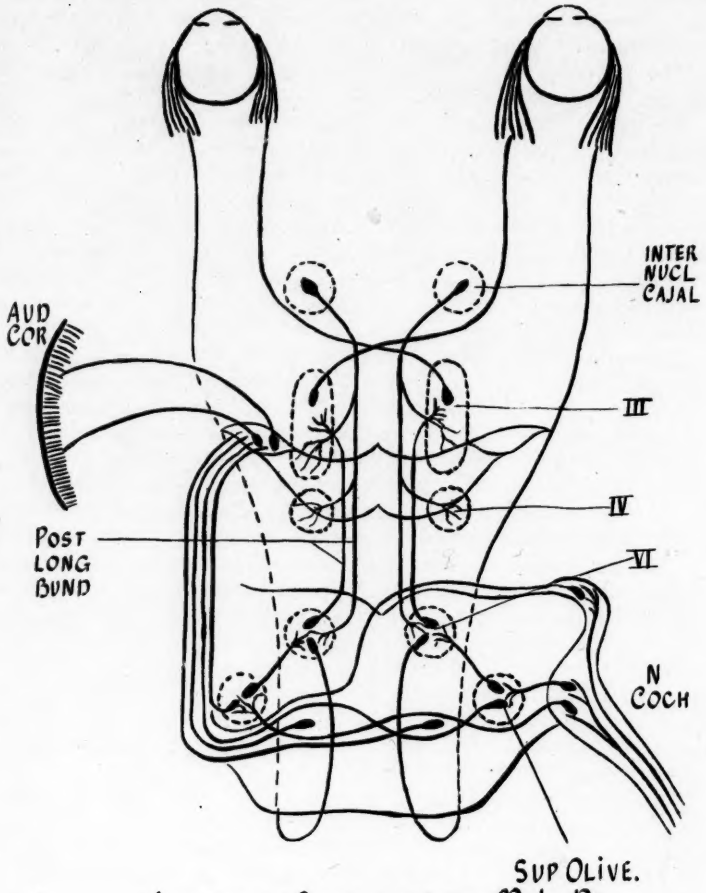
The fibres arising from the fourth nucleus, unlike those arising from the posterior part of the third nerve, do not cross before emergence from the brain substance, but pass first obliquely downwards, and then turn dorsalwards, and emerging from the brain substance, cross in the velum medullare in the anterior part of the roof of the fourth ventricle.

The nerve fibres from the sixth nucleus do not cross, but pass ventrally to supply the external rectus of the same side. Obersteiner has described fibres crossing the raphe between the two sixth nuclei, but it is by no means certain that these fibres are related to the sixth nuclei, since at this position, the dorsal longitudinal bundle lies between the cell groups and the middle line, and the fibres crossing the raphe at this level may be from the dorsal longitudinal bundle, or from Deiter's nucleus, crossing to enter the dorsal longitudinal bundle of the opposite side.

The three nuclei are all intimately linked up with one another, and with lower centres, by bundles of longitudinal fibres, and so important are these linking fibres that Lhermitte[9] has suggested the use of the term "internuclear paralysis" to distinguish the important forms of paralyses that may occur from lesions in the course of these fibres, and differentiate them from supranuclear, nuclear, and infranuclear paralyses. Of the connections of the dorsal longitudinal bundle of Schütz which lies dorsal to the oculomotor nuclei in the ependymal grey matter surrounding the aqueduct of Sylvius, I must confess my ignorance. Schütz supposed it to be a connecting link between the oculomotor nuclei, but it has been traced into a nucleus, the dorsal tegmental nucleus of Gudden, in front of the oculomotor area, and backwards, still in the ependymal tissue, in the floor of the fourth ventricle, to the pons and medulla. It is presumed to be a primitive tract linking up the olfactory system with the oculomotor nuclei and lower motor nuclei.*[10 and 11]

The most important bundle of internuncial fibres linking up the oculomotor nuclei is the posterior longitudinal bundle. This may be described as a composite bundle composed of axones arising in different groups of cells. It contains both ascending and descending fibres. It can be traced right down the cord in the anterior column, linking up with anterior horn cells, and anteriorly it can be traced forwards into the subthalamic region, and recently Fraser has traced it into the thalamus. Its anterior connections are somewhat obscure, but it receives fibres from the nucleus of Darkschewitsch, which brings it into relationship with the posterior

*_Vide_ Addendum, Wherry's case, _T.O.S._, Vol. XI, p. 118.



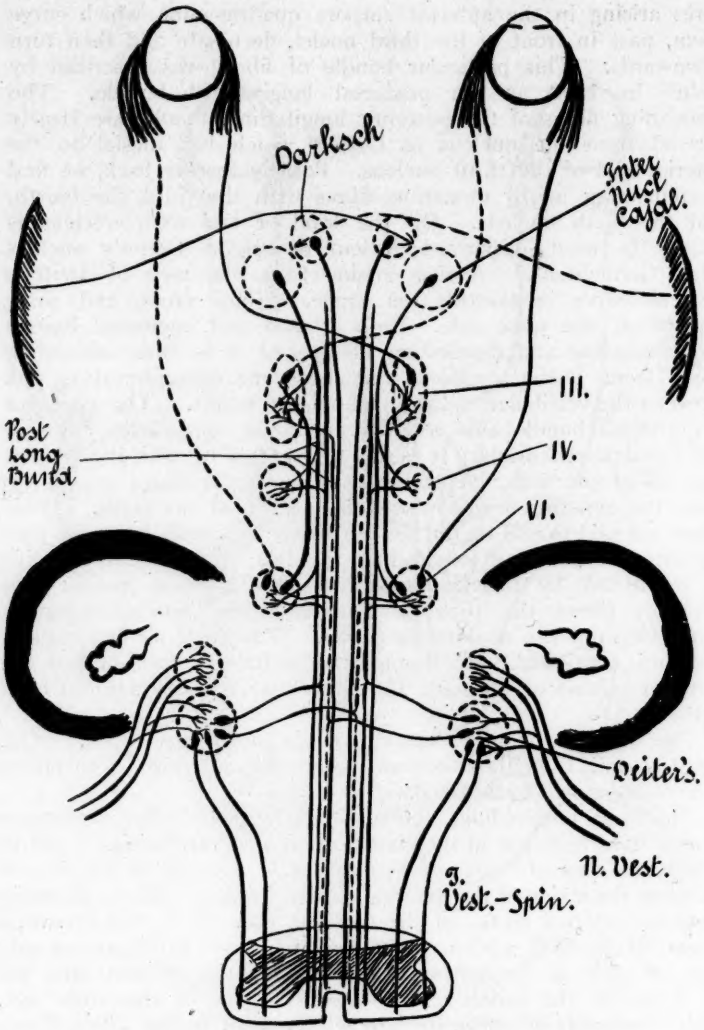
AUDITORY CONNECTIONS P. L. B.

From Villiger, "Brain and Spinal Cord," 1912.

commissure. The ventral portion of the bundle is formed from fibres arising in the anterior corpora quadrigemina which curve down, pass in front of the third nuclei, decussate and then turn downwards. This particular bundle of fibres was described by Held[12] in 1890 as the predorsal longitudinal bundle. The descending fibres of the posterior longitudinal bundle are largely derived from the nucleus of Cajal,[10] which lies mesial to the anterior end of the third nucleus. Passing further back, we find it connecting up by numerous fibres with the third, the fourth, and the sixth nucleus. On the level of the sixth nucleus is found its most important nucleus of origin—Deiter's nucleus (Van Gehuchten).[28] Axones arising from the cells of Deiter's nucleus curve in towards the raphe. Some cross, and some remain on the same side. Both crossed and uncrossed branch into ascending and descending fibres, and it is these ascending fibres from Deiter's nucleus that form the communicating link between the vestibular and the oculomotor nuclei. The posterior longitudinal bundle also receives numerous fibres from cells in the formatio reticularis. It is said also to link up with the sensory nucleus of the fifth. A well-marked bundle of fibres sweeps up from the superior olive towards the nucleus of the sixth. These fibres are said to end in the sixth nucleus, but some probably pass up into the posterior longitudinal bundle. As the superior olive is connected by the trapezium with the auditory nuclei, this probably forms the path of communication between auditory sensations and the oculomotor nuclei. The posterior longitudinal bundle is continued down through the medulla to form part of the anterior column of the cord and thus links up with anterior horn cells.

The diagrams from Villiger's[13] "Brain and Spinal Cord," 1912, pp. 182 and 183, illustrate some of these important connections I have attempted to describe.

The dorsal longitudinal bundle is of such great clinical importance that I have ventured in the classification of ocular palsies to put in a separate class of "inter-nuclear palsies." One of its functions is to bring the action of the external rectus of one eye into relationship with the internal rectus of the opposite eye. As is well known, a lesion in the sixth nucleus or its neighbourhood produces not only loss of outward movement of the eye on the affected side, but a defect in the action of the internal rectus of the other eye, only observable in conjugate lateral movement to the affected side and not present in convergence. Duval and Laborde[14] suggested that fibres originating in the sixth nucleus pass up the dorsal longitudinal bundle, enter the third nucleus and cross to the nucleus of the opposite side, and pass out with the third nerve to the right internal rectus. Neither von Gudden nor von Koelliker have



Vestibular Connections. P. L. B.

From Villiger, "Brain and Spinal Cord," 1912 (modified).

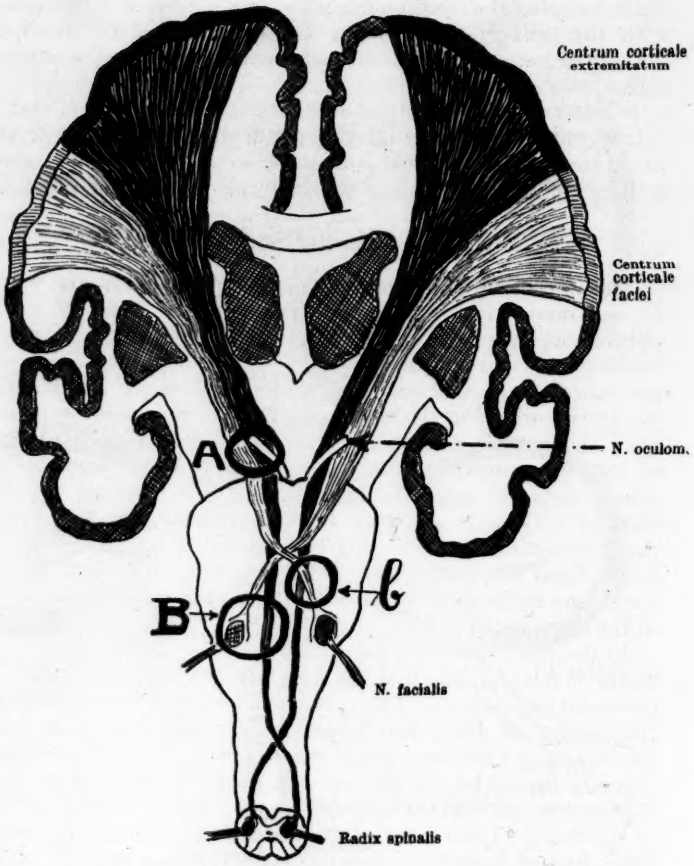
been able to find any proof of this assumption, and it seems more probable, from other associated ocular movements, that the internal recti are supplied partly by direct fibres from the nucleus of the same side, and partly by crossed fibres from the opposite nucleus. In which case, the longtitudinal bundle will serve as a link in the chain bringing the sixth nucleus of one side into communication with the cells of origin of the crossed fibres of the third, either directly or more probably through the interposition of intercalary cells.

It must not be forgotten, as Sherrington[1] has shown, that every active contraction of the internal rectus implies an active relaxation of its antagonistic external, and these inhibitory impulses also may well be communicated along the posterior-longitudinal bundle.

Vascular Supply

The important part that vascular lesions play in the causation of oculomotor paralyses is well recognised. Many of the acute ophthalmoplegias occurring in infective or toxic diseases are the result of vascular changes set up in these diseases. But we also get more direct vascular lesions resulting from the bursting of miliary aneurisms in the smaller cerebral vessels, and haemorrhages from the smaller arteries in arterio-sclerosis, and in other forms of local or general arteritis. Furthermore, the oculomotor nuclei, nerve roots, or association fibres, may be involved in areas of cerebral softening, resulting from thrombosis, or the nerves in their intracranial course may be pressed on by aneurisms in the larger basal vessels, and the third, and to a less extent, the fourth nerves are liable to be affected by thickening of the internal carotid in the cavernous sinus.

In the first place, it is important to realise that the area of oculomotor nuclei is supplied by end arteries and that there is no collateral circulation. These end arteries come either directly from the basilar or from the posterior cerebral, very soon after its bifurcation. Clinically, there seems much evidence pointing to a separate supply for the anterior and posterior parts of the nucleus. The vessels run dorsally upwards on the inner side of the bifurcation of the crura. Leon d'Astros and Alezais[15] sub-divide the supply into three lots of branches arising from one artery from the posterior cerebral, an anterior set passing horizontally forwards to the anterior end of the third nucleus; a middle set passing vertically; and a posterior set passing obliquely to the posterior end of the third and the fourth nucleus. Bernheimer[3] claims to have traced a separate supply to the Edinger-Westphal nucleus from the dorso-lateral region of the crura, a branch from the vessel supplying the anterior corpus quadrigeminum.



From Bing "Kompendium der topischen Gehirn und Rückenmarks Diagnostik," 2nd Edit., 1911.

Very few cases of limited or partial affections of the oculomotor nuclei from vascular lesions have been examined pathologically, and in the few autopsies made, the interpretation has been confused by the fact that nerve roots, as well as cells, have been involved in the affected area.

In the great majority of cases of haemorrhage or softening affecting this region, the crura cerebri, the mid brain and the pons are the main sufferers. The resulting ophthalmoplegia is often more properly a nerve root lesion than a nuclear lesion. These form the typical crossed hemiplegias.

The interest of these crossed hemiplegias may be my excuse for digressing for a few moments from my main subject. I have copied a diagram from Bing[16] to illustrate some of the principal forms. They may result from any form of lesion, vascular, tuberculous, gummatous, neoplastic or inflammatory, affecting the pons or the cerebral peduncles.

Commencing from above, we get the typical "Weber's syndrome," a complete paralysis of one third nerve, with a hemiplegia of the opposite side. This lesion must be at the posterior end of the crus or the anterior end of the pons, and the roots of the third are involved on their way out.

In "Benedict's syndrome," there is oculomotor paralysis on one side, with tremors and convulsive movements of the opposite arm, or opposite limbs.*

The lesion in this type lies deeper in the mid brain and the nucleus ruber has been found to be markedly affected in such cases and consequently, the root fibres of the third, as they run through it.

In "Foville's syndrome," the lesion is situated lower down. The nucleus of the sixth is affected, and in addition to paralysis of the external rectus on the same side, there is loss of the conjugate deviating action of the internal rectus of the eye on the opposite side with hemiplegia of the opposite side, so that the eyes are turned towards the side of the paralysed limbs. There is facial paralysis on the side of the paralysed external rectus.

In the "Millard-Gubler syndrome," there is complete paralysis of the sixth and seventh on one side, with crossed hemiplegia, but the movements of the opposite internal rectus are unaffected. In this case, the lesion is near the posterior end of the pons, and the sixth root is affected, but not the sixth nucleus.

I have tried to simplify my account of these complex conditions by leaving out the intricacies of the fifth nerve roots, and their participation in these lesions.

**Vide* Addendum *T.O.S.*, Lees, Vol. III, p. 278, p. 281. Taylor, Vol. XVIII, p. 381. Jessop, Vol. XXIX, p. 271. Batten, Vol. XXX, p. 116.

PART II.—CLINICAL

In the first part of my paper I have attempted to define an area of the central nervous system within which disease is almost certain to set up some derangement of the oculomotor system, and before going further, I think it is necessary to point out that there can be no sharply defined limits between lesions affecting nuclei, *i.e.*, cell groups, nerve roots, or association fibres, and this is specially the case where the lesion is primarily or secondarily of vascular origin. Mauthner[17] defined an ophthalmoplegia as a condition in which either muscles of both eyes are affected, especially if they are supplied by different nerves, or if the movements of one eye only are affected, more than one nerve of supply must show evidence of affection. This especially includes cases where all the eye muscles are affected—complete ophthalmoplegia—and also where all the external muscles are affected, and the sphincters of the iris and the ciliary muscles escape—ophthalmoplegia externa. Uhthoff definitely excludes from his discussion of the ophthalmoplegias double external rectus palsies and double isolated ptosis, on the grounds that though they are often undoubtedly of nuclear origin, they can also be due to other causes. I think it is further desirable to limit the discussion by excluding cases of pure ophthalmoplegia interna.

Classification of Ophthalmoplegias

I. *Acute and sub-acute Ophthalmoplegias.*

(A) *Poisons.*
Alcohol.
Lead.
Botulism.
(Carbon monoxide; nicotine; snake poison; sulphuric acid.)

(B) *Infective agents.*
Diphtheria.
Influenza.
Lethargic encephalitis.
Syphilis.
Tubercle.
(Measles, typhus, chicken-pox, septicaemia, chills.)

(C) *Trauma.*

(D) *No etiological factor.*

II. *Chronic* (Progressive) Ophthalmoplegias.

(A) An isolated lesion.

(B) Tabes and General Paralysis.

(C) Disseminated Sclerosis.
Posterolateral sclerosis (sub-acute combined.)
Syringomyelia. Paralysis Agitans.

(D) Bulbar Palsies. Progressive muscular atrophy. Myasthenia Gravis.
(E) Graves' Disease.
(F) Diabetes.

III. Congenital Ophthalmoplegias of nuclear origin.

IV. Unilateral Ophthalmoplegia.

V. Recurrent and alternating Ophthalmoplegia of nuclear origin.

Acute Toxic Ophthalmoplegia

Of the toxic forms of acute ophthalmoplegia, that due to alcoholic poisoning may be taken as the type. This was first described by Wernicke[18] in 1881, under the name of acute haemorrhagic polioencephalitis superior. In its most typical manifestation, it shows a rapidly developing ophthalmoplegia, sometimes affecting the pupils and sometimes accompanied by ptosis. The general condition renders it difficult to examine accommodation. The onset is accompanied often by somnolence, or there may be acute delirium. There is severe headache and giddiness. Bulbar nuclei become implicated and speech may be affected. The gait may become ataxic. Knee jerks may be lost, and other deep reflexes be affected, weakness may develop in all limbs, and death may follow in from ten to fourteen days. Not all the cases, however, end fatally, and in some which do so end, death does not take place for some time.

Autopsy shows numerous punctiform haemorrhages in the grey matter in the walls of the third ventricle, below the aqueduct of Sylvius and in the floor of the fourth ventricle. The vessels show fatty and sclerotic degeneration and cellular infiltration in their sheaths. The nerve stems at their exit from the brain are usually found to be healthy.

In lead poisoning, the majority of cases of oculomotor disturbance are probably due to a neuritis. In some cases, however, there is evidence of a nuclear origin, but these do not show the numerous other severe and acute cerebral complications found in alcoholism.

The clinical picture produced in botulism also differs in many respects from the Wernicke type. In practically every case, both pupils and ciliary muscles are paralysed, and in more than 50 per cent. of the cases, there is more or less pronounced double ptosis. The other ocular muscles may be affected in a varying degree. The general symptoms of a food poisoning usually precede the onset of the ocular symptoms, and point to the diagnosis. Experimentally, von Emengen, Marinesco and others have shown that degenerative changes take place in the grey matter in the region of the oculomotor nuclei.

Acute Infective Ophthalmoplegia

I think I may be excused if, at the present time, I take lethargic encephalitis as an example of infective ophthalmoplegias. In its most typical manisfestations, the disease begins with a disturbance of oculomotor functions. Accommodation is almost invariably affected, the pupil very frequently, and in certain cases, a typical Argyll Robertson pupil has been described. The other ocular movements may be affected in such various ways as to suggest that the lesions may be nuclear, infranuclear, or supranuclear.

The marked somnolence which occurs in the disease is, as I have already mentioned, met with also in the alcoholic type of Wernicke, and as Mauthner showed many years ago, is a characteristic of various lesions affecting the region of the posterior end of the third ventricle. It is met with not infrequently in pituitary tumours, and Salmon has suggested that in these cases, it is due to disordered pituitary secretions. Marie and Trétiakoff have demonstrated, however, the integrity of the hypophysis in some cases of encephalitis where somnolence was a marked feature.

It is of interest to note how the onset of normal physiological sleep may be accompanied by signs of loss of activity of various ocular muscles. Many of you in reading may have noted how the print of the book may seem to diminish in size, or the objects in a room look as if seen through the wrong end of a telescope, a micropsia due to the increased effort necessary to maintain a failing accommodation. The increase in size of the pupils is common knowledge, and also the tendency of the words to blur and run into one another, owing to failing convergence power. Later comes on the ptosis of sleep, and with it, the unconsciousness. There seems to be little doubt that pathological somnolence may arise in the same way from disturbances in the neighbourhood of the third nuclei.

It is not within my capacity to discuss the symptoms resulting from the spread of the changes in encephalitis lethargica into other parts of the basal grey matter, into the cortex, and even into the medulla and spinal cord.

Macroscopically, the grey matter in the mid brain is seen to be studded with small haemorrhages, and there seems little doubt that the favourite site of this affection is in the grey matter in the region of the aqueduct of Sylvius, and however widespread the lesions may be, they are often at their maximum in this neighbourhood. (Locus Niger. Pierre Marie and Trétiakoff.)

Microscopically,[9] the lesions may be seen to be of three kinds :— (1) A dense perivascular infiltration into the Virchow-Robin space. The cells composing this infiltrate are mononuclear lymphocytes, plasma cells of Unna, polyblasts and polynuclear neutrophils. Many

vessels also show this space filled with red corpuscles, either as the result of rupture, or, according to Economo, from diapedesis, and according to Economo, the extravasation of red corpuscles is restricted to grey matter, and does not take place where the vessels enter white matter. There is little doubt, however, that this is too narrow a view. The condition in many cases, as von Monakow has shown, may lead to more or less extensive thrombosis, not necessarily restricted to intracerebral veins, and this thrombosis itself may be the cause of some of the haemorrhages.

(2) The second type of lesion found is in the form of inflammatory nodules in the tissues away from the vessels, the cells in these being very similar in type to those found in the perivascular lesions.

(3) The third type of change is a phagocytosis of nerve cells, a condition called "neuronophagia."

It is quite obvious from lesions so extensive and so profound as are to be found in this disease that, though the site of election of the disease may be the nuclei, the commissural fibres and the nerve roots cannot escape being affected, and from the mode in which it spreads, we are justified in expecting to find supranuclear and infranuclear lesions, in addition to the more typical nuclear lesions.

As to the causative organism, the evidence at present seems to point to its being a filtrable organism described by Hervier and Levaditi, rather than the gram-positive non-filtering organism described earlier by Wiesner.*

Of other causes of acute infective polioencephalitis, influenza is probably the most important. Cases have been recorded showing the most varied extent of involvement of the extrinsic eye muscles. The pupils are sometimes affected and more frequently accommodation. While there is undoubted evidence that influenza can set up nuclear lesion, it may also at times cause ocular palsies from basal affections, or from a peri-neuritis.

The evidence as to the nature and site of the lesion causing oculomotor paralyses in diphtheria is very contradictory. According to Babinski, Enriques and Hallion, Muravieff and others, there is a primary affection of nuclei, and this is sometimes associated with a similar affection of anterior horn cells with secondary degenerative changes in the peripheral nerves.

Bielschowsky, Sidney Martin and others regard the nerve change as the primary one, a parenchymatous neuritis, and look on the changes in the nuclei and anterior horn cells as secondary. In the majority of diphtheria cases where there is an ocular palsy, it is only accommodation that is affected, and when, as is very frequently the case, the soft palate is also paralysed, the diagnosis of a diphtheritic

* Cases of Lethargic Encephalitis, *vide* Addendum, *T.O.S.*, Vol. XXXVIII, Taylor, p. 168. Batten, p. 172.

cause for the condition is much strengthened. Personally, the only extrinsic eye muscles I have ever seen affected after diphtheria are the external recti, and they are said to be affected in about 10 per cent. of cases showing ocular palsies. It is only very rarely that other eye muscles are affected.

In some rare cases, injuries, especially blows on the head without any evidence of basal fracture, have produced ophthalmoplegias of nuclear origin.

There remains a very definite percentage of cases of acute or sub-acute ophthalmoplegia in which no definite etiological factor is discoverable. In Uhthoff's statistics, 33 per cent. of the cases fell into this category. At present, many of these would be, and are, classified as cases of lethargic encephalitis, but in course of time, a better sense of proportion will supervene, and when it ceases to be the "nine days wonder" of the Daily Press, we may again recognise that not all cases of acute polioencephalitis are to be lumped together under that very convenient heading. The cases in which no etiological factor is discoverable may be classified in two groups:—(1) Those without other complications, and (2) those showing evidence of lesions in the lower part of the bulb. Probably many of the latter are cases of lethargic encephalitis. The first group is the larger, and a definite proportion of them are probably vascular in origin.

The Chronic Progressive Ophthalmoplegias.

In an earlier part of the paper, I have referred to the ophthalmoplegias which occur in association with vascular lesions affecting the crura cerebri and pons. A certain proportion of chronic ocular palsies is associated with evidences of patches of softening affecting these regions. But the diseases which bulk most largely in the causation of chronic progressive ophthalmoplegia are undoubtedly tabes and general paralysis.

(1) *As an isolated lesion.* Before dealing with these cases, however, it is desirable to point out that a form of chronic ocular palsy occurs without any sign of further affection of the central nervous system. It occurs mostly in comparatively young people, though in the case recorded by Lawford (*Trans. Oph. Soc.*, Vol. VII., p. 260), the age at commencement was fifty, and one other case has been recorded commencing at fifty-three. In these cases, there may be complete ophthalmoplegia externa, with ptosis in both eyes, but neither pupils nor accommodation are affected. In this respect, Lawford's case was typical. This type occurs as a definite hereditary affection, *vide* papers by Beaumont and Bradburne in addendum.*

*T. O. S., Beaumont, Vol. XX, p. 258. Bradburne, Vol. XXXII, p. 142

(2) *In tabes and general paralysis.* According to Uhthoff's[19] statistics, about 20 per cent. of all cases of ocular palsies occur in association with tabes or general paralysis, and Wilbrand and Saenger[20] found in 302 cases of tabes 22 cases of ophthalmoplegia (7·2 per cent.). We must remember in these cases that we are dealing with cases the majority of which are syphilitic and that quite apart from tabes, syphilis can produce an ocular palsy in many different ways; but with all allowances, there is no doubt that tabes, and to a less extent, general paralysis, are very common causes of ophthalmoplegia. There seems to be little doubt that the primary changes take place in the cells of the nuclei, and that as time goes on, the whole neuron from the nucleus down to the muscle (and in the end, the muscle itself) becomes affected. The changes have been described in detail by Siemerling and Boedeker[21]. The cells are small and shrunken, or even completely degenerated, or they show vacuolation. The cell processes have disappeared and the nucleus and nucleolus are degenerate. There is definite glial overgrowth. The amount of nerve degeneration is proportional to the degree of cell degeneration shown. In the later stages, the degeneration reaches the muscle, and the same muscle may show some fibres completely atrophied, and others grossly hypertrophied and a great increase of interstitial connective tissue.

The fleeting diplopias and ocular palsies of early tabes have probably quite another explanation, and should not, I think, be regarded as early stages of a nuclear degeneration. Sherrington[1] has made the interesting suggestion that these may be caused by an affection of the afferent proprioceptive fibres, which have been shown to exist in the oculomotor nerves.

(3) In disseminated sclerosis, temporary diplopias and temporary disorders of associated ocular movements are comparatively frequent, while permanent progressive ophthalmoplegia is one of the rarer manifestations of the disease. Probably quite a number of the disorders of eye movements which do not result in diplopia and are of a fleeting character, are missed or forgotten, when the more classic symptoms of disseminated sclerosis become manifest. I have seen cases in which two or three attacks of temporary diplopia at intervals of some years have occurred before any more definite signs of the disease have been shown. The clinical signs point to an interference with nerve roots or commissural fibres, rather than a nuclear palsy, and this is in consonance with what is known of the pathology of the disease. Similarly in the rarer forms of permanent ophthalmoplegia occurring in disseminated sclerosis, the lesion is probably infranuclear and due to plaques occurring in the pons or crura.

(4) We next come to a very definite group of cases where ophthalmoplegia is combined with a bulbar palsy or a progressive muscular atrophy, or where all three are present in combination.

According to Uhthoff,[19] about 19 per cent of the cases of chronic progressive ophthalmoplegia fall into this group, and in about half of these, the combination is one of ocular and bulbar palsies.

Chronic ocular palsies have been recorded in Graves' Disease, and in diabetes, but in the latter disease, we probably have to do much more frequently with a peripheral neuritis than with a palsy of nuclear or root origin.

Congenital Ophthalmoplegias

Congenital defects of ocular movements form by themselves a subject which might occupy the whole of our time. They can be divided into three different clinical groups :—

(1) The defective ocular movement is an isolated and non-progressive phenomenon.
(2) The defect is hereditary, and may be accompanied by other defects of an hereditary nature.
(3) Other elements of the central nervous system may show analogous congenital defects.

Pathologically, any part of the oculomotor system may be affected, so that at the one end we may get a complete aplasia of the oculomotor nucleus and at the other end all sorts of peculiarities in the development of the external musculature of the bulb.

In conclusion, I must acknowledge my indebtedness to various neurological friends for aid in looking up references, and to Professor Uhthoff's excellent account in Vol. XI of Graefe-Saemisch "Handbuch der Gesamten Augenheilkunde," and Volumes I and VIII of Wilbrand and Saenger's "Neurologie des Auges."

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ADDENDUM

Cases and Papers in the *Transactions of the Ophthalmological Society* on Ophthalmoplegia.

VOL. I.

Gowers, p. 117. "Paralysis of upward movements with optic neuritis." Both eyes.

Jackson, p. 139. "Eye symptoms in locomotor ataxy."

Nettleship, p. 164. "Double isolated ophthalmoplegia externa." Youth, aged 18. Pupils and accommodation unaffected.

Sturge, p. 165. "Two cases of simultaneous paralysis of both third nerves." First case undoubtedly due to haemorrhage. Second case, probably a haemorrhage occurring in association with some inflammatory or neoplastic lesion.

Benson, p. 265. "Ocular paralysis after diphtheria." Accommodation and soft palate affected, also ptosis and paralysis of external recti.

VOL. III.

Lees, p. 278. "Paralysis of right third with cerebral symptoms" A case of right third nerve paralysis with marked tremor in left arm and leg. Probably lesion in mid brain. No note of facial weakness; *v.* Taylor's case, Vol. XVIII and Batten's case, Vol. XXX.

Lees, p. 281. "Paralysis of left sixth with spasmodic contractions of right face."

VOL. IV.

Eales, p. 300. "Complete paralysis of accommodation and convergence." A girl, aged 13. No other nervous system lesion. Pupils active to light.

VOL. V.

Sharkey, p. 384. "Locomotor ataxy with ophthalmoplegia externa and interna."

Jessop, p. 386. "Diphtheritic cycloplegia, with absence of knee jerks, etc." Loss of accommodation. Pupils active. No extrinsic paralysis of eye muscles. Soft palate affected. Knee jerks absent. Visual fields affected.

VOL. VII.

Coupland, p. 228. "Ophthalmoplegia dependent upon thrombosis of cavernous sinus."

Robinson and Hutchinson, p. 250. "Ophthalmoplegia, externa and interna." Disease of right cavernous sinus resulting in its obliteration. Syphilitic origin. A discussion of some other cases of syphilitic ophthalmoplegia both of central and peripheral origin.

Lawford, p. 260. "Double ophthalmoplegia externa without other symptoms." Referred to in paper; *v.* also Beaumont, Vol. XX, p. 238.

Gowers, p. 269. "Conjugate ocular palsy and nystagmus." A discussion of the lesions in the neighbourhood of the sixth nucleus which cause conjugate palsies; i.e., paralysis of lateral movement of opposite internal rectus with full retention of movement on convergence.

Beevor, p. 281. "Ophthalmoplegia externa without other symptoms." Commencing in a woman at age of 34. Pupils and accommodation unaffected.

Mackinlay, p. 281. "Paralysis of both external recti, probably congenital."

VOL. VIII.

Lawford, p. 262. "Congenital hereditary defect of ocular movements." A family in which the father, one son, and two daughters showed similar defects in ocular movements. Quotes Heuck's paper (*Klin. Monatsbl. für Augenheilk.*) in which a similar family is described and autopsy in one case showed the defects to be due to muscular anomalies.

VOL. XI.

Stanford Morton, p. 106. "Bilateral paralysis of external recti following diphtheria." Four cases showing paralysis of external recti. Interesting feature of one case was that accommodation did not seem to be affected. Pupils were normal. Soft palate affected. Knee jerks absent in all cases.

Wherry, p. 118. "Ophthalmoplegia associated with parageusia."

VOL. XIII.

Snell, p. 110. "Two cases of recurrent third nerve palsy with migraine." Typical ophthalmoplegic migraine. In one case the ophthalmoplegia had become permanent.

VOL. XV.

Beevor, p. 242. "Ophthalmoplegia externa without other symptoms." Woman. Symptoms commenced at age of 25. Pupils and accommodation not affected. *v.* also Vol. VII, page 281.

VOL. XVI.

Spicer and Ormerod, p. 277. "Recurrent paralysis of ocular nerves." Seven cases of typical ophthalmoplegic migraine: with a full discussion of the previously published cases, and the nature of the lesion causing the phenomena.

Grimsdale, p. 298. "Recurrent paralysis of right third nerve."

Waren Tay, p. 302. "Double ptosis with loss of convergence and weakness of internal recti."

VOL. XVIII.

James Taylor, p. 381. Ophthalmoplegia externa with bulbar symptoms and marked weakness of orbicularis palpebrarum on each side," Diplopia, ptosis, weakness of left arm. Definite bulbar symptoms. Weakness of orbicularis on both sides. *v.* also Lees, Vol. III; Jessop, Vol. XXIX; and Batten, Vol. XXX.

VOL. XX.

Beaumont, p. 258. "Family tendency to ophthalmoplegia externa." Eleven members of a family in four generations showing varying degrees of ophthalmoplegia externa. Interesting, as it includes Lawford's case, *v.* Vol. VII. Most of the cases began in early adult life.

VOL. XXI.

Berry, p. 268. "On so-called paresis of divergence." A very full and lucid argument against the existence of special divergence innervation and a centre for divergence.

VOL. XXII.

Harman, p. 280. "Paresis of third nerve, in which, on lifting the drooping lid there is coincident drooping of the sound lid."

Spicer, p. 304. "Tabetic paresis of convergence with Argyll Robertson pupils."

VOL. XXIII.

Bishop Harman, p. 356. "Innervation of the orbicularis palpebrarum muscle." A discussion of the hypothesis of Mendel that the portion of the facial nerve concerned in innervation of the orbicularis palpebrarum has its nuclear origin in the oculo-motor nucleus and reaches the facial by the posterior longitudinal bundle. Harman on morphological grounds is strongly opposed to Mendel's hypothesis.

VOL. XXVII.

Ludford Cooper, p. 182. "Paralysis of vertical movements of both eyes. (? Haemorrhage about the corpora quadrigemina)." (No note of pupil reactions).

VOL. XXIX.

Jessop, p. 271. "Bilateral ptosis and ophthalmoplegia externa." A child, aged 5, showed rapidly developing ophthalmoplegia externa with ptosis. Later symptoms

of bulbar involvement came on. Death four months later. No P.M. Probably a tuberculous process commencing in the mid brain; *v.* Lees, Vol. III; Taylor, Vol. XVIII; Batten, Vol. XXX.

VOL. XXX.

Batten, p. 116. "Bilateral ophthalmoplegia externa in an infant." A case resembling Jessop's, *vide supra*. The child showed also weakness of right arm and leg, with constant rhythmic movements, and subsequent convulsions on right side. Death after 6 months. P.M., a large tuberculous mass involving optic thalamus and corpora quadrigemina with a second tuberculous mass on the surface of left occipital cortex.

VOL. XXXII.

McMullen, p. 110. "External ophthalmoplegia of long standing." Commenced at age of 8. Pupils and accommodation normal.

Bradburne, p. 142. "Hereditary ophthalmoplegia in five generations." Sixteen out of thirty-seven individuals affected in five generations. In most of the cases all the external muscles were affected and ptosis was present in some but not all the cases. In the few cases where it is noted, pupillary movements and accommodation seem to have escaped.

VOL. XXXIII.

Cunningham, p. 156. "Paresis of both internal recti." Probably belongs to the class of case where a lesion in the posterior longitudinal bundle interferes with lateral deviation. Movements on convergence were not so markedly affected.

VOL. XXXIV.

Coats, p. 262. "Congenital partial paralysis of the oculo-motor nerve with cyclical contraction and dilatation of the pupil." A case in which rhythmical action of the pupil of the left eye accompanied a partial paralysis of branches of the left third. Partial ptosis. Complete loss of upward movement. Inward and downward movements defective.

VOL. XXXVIII.

James Taylor, p. 168. "Paralysis of ocular muscles." Occurring in: 1, myasthenia gravis; 2, Grave's disease; 3, lethargic encephalitis.

F. E. Batten, p. 172. "Cases of lethargic encephalitis." These were some of the earliest cases of lethargic encephalitis shown in this country.

VOL. XL.

Cross, p. 277. "The nerve paths and centres concerned in sight." A very full account of the anatomical arrangement of the nerve centres and tracts including the comparative anatomy.

ANNOTATIONS

Swanley Ophthalmia School and Non-Pauper Children

It has often been said that the British Constitution grew and was not made; this statement might also be well applied to the arrangements for dealing with infectious and contagious diseases in the Metropolis.

The Metropolitan Asylums Board was established in 1867, by an order of what was then called the Poor Law Board, for the reception and relief of poor persons chargeable to some union or parish of London, infected with fever or smallpox, or suffering from insanity. The Poor Law Board subsequently became the Local Government Board, and in 1883 the Disease Prevention Act (London) removed the civil disabilities which had till then been attached to admission into the Metropolitan Asylums Boards Hospitals.

The Poor Law Act, 1889, empowered the Board to admit non-pauper cases of fever, diphtheria, and smallpox.

In 1911 the Local Government Board sanctioned the admission to any of the infectious hospitals of poor persons suffering from such infectious or contagious disease other than those already mentioned, as they might thereafter determine. In accordance with this order they in 1912 sanctioned the admission of children suffering from measles and whooping cough, and later in the same year, subject to certain restrictions, the admission of non-pauper patients suffering from those diseases.

By an order issued in 1897 the Local Government Board constituted the Metropolitan Asylums Board the central authority in the Metropolis for dealing with various classes of Poor Law children, amongst them those suffering from ophthalmia. As the outcome of this order, the Metropolitan Asylums Board in 1903-4 opened two schools, one at Swanley and the other at Brentwood, for the isolation, treatment, and education of Poor Law children suffering from contagious eye diseases. These schools provided accommodation for about 600 children and up to 1910 it proved barely sufficient for those sent to them for treatment. Since that date there has been a gradual and steady decline in the number of children affected with contagious eye diseases in the London Poor Law schools, due mainly to the isolation which the ophthalmia schools provided checking the spread of those diseases. As the result of this decrease it became possible to give up the Brentwood School as an ophthalmia school, and for some time now the Swanley School has only been partly occupied.

The only children eligible for admission to these ophthalmic institutions were those whose parents were in receipt of Poor Law relief. The children of parents who were more thrifty and who struggled against the acceptance of anything implying the taint of pauperism, if they contracted ophthalmia, became excluded from attendance at their day schools, had to attend frequently at hospitals for treatment, and owing to the absence of proper home attention and suitable hygienic surroundings, often dragged on for months and months a miserable existence.

In the summer of last year the Council of British Ophthalmologists, impressed by the number of children in London suffering from contagious eye disease. They drew up a report advocating the admission of non-pauper children suffering from contagious eye diseases into the Metropolitan Asylums Board Ophthalmia School. This they forwarded to the Minister of Health asking him to receive a deputation in order that they might lay the views of the Council more fully before him.

This request met with a cordial reception and in due course a deputation was received by Sir George Newman, who, after the

members of the Council had presented their case, expressed himself as in complete sympathy with the object it had in view, and promised to do what he could to get non-pauper patients made eligible for admission to the Metropolitan Asylums Board's ophthalmia institution as had been done for admission to their fever hospitals.

The necessary negotiations and arrangements have been carried out, and now any child attending at a London County Council school, suffering from a contagious eye disease, can be admitted to the ophthalmia school at Swanley, provided accommodation for the class of children for which it was originally intended is not encroached upon.

The Council of British Ophthalmologists may be congratulated upon the success of their intervention in this matter, calculated as it is, not only to restore numerous poor children to health and efficiency, but also to be a benefit to the community by checking the spread of contagious maladies of the eye amongst its members.

The Preparation of Scientific Papers

Those who aspire to write for the medical press have been placed under a debt of gratitude by the Medical Research Council, the publications department of which has recently (January, 1921) issued a brochure entitled "Notes upon the Preparation of Monographs and Reports for Publication by the Council." It embodies much information likely to be useful to such writers, and not easy to come at by the usual channels. The primary object of the pamphlet appears to be to keep the cost of publications and reports down to the lowest possible figure, a precaution rendered the more necessary on account of the rapidly increasing cost of printing. Care should be taken that copy be typewritten, or, when that is not possible, the utmost care should be taken that proper names and titles should be written legibly. In any case, one side only of the paper should be used, and the MS. should be sent packed flat and not rolled or folded. An author should correct his typewritten copy before it leaves his hands. Reference lists, tables, etc., should be sent in on separate sheets of paper, which, so far as may be, should be of uniform size. When quotations are inserted in the text, it is suggested that, if more than four lines long, they should be marked by a vertical line placed on the left-hand side to indicate that they are to be printed in a separate type. New paragraphs should be indicated by indenting the first line of the new paragraph about two inches. Footnotes, as a rule, are not desirable. The best method of keeping and arranging references is by the so-called "Harvard System." (1) According to this plan, all works cited are collected together at the end of an article where they are arranged in alpha-

betical order according to authors' names, followed by initial and printed in small capitals. (2) This is followed by the date of publication of the work enclosed in brackets. If more than one paper by an author has to be cited for the same year the different papers are distinguished by adding immediately after the date the letters a, b, c, etc. to each. (3) Full title of paper or work itself in Roman type. (4) In writing the title of a journal or other periodical publication the abbreviations employed in the catalogue of the Library in the Surgeon-General's office, United States Army, should be used and such abbreviations be printed in italics. (5) The volume of the journal or other periodical is indicated by an Arabic numeral in clarendon type. (6) The page on which the article begins is indicated by an Arabic numeral. Works by the same author are arranged in chronological order. Obscure or misleading abbreviations should be avoided. A list of abbreviations for units of the metric system is given. If there be any possibility of doubt, "gramme" and "grain" should be spelt in full and no attempt be made to abbreviate them. Where degrees of temperature are referred to, the scale employed should always be specified. To indicate a percentage 'per cent.' should be used and not the symbol %. The familiar caution is repeated, namely, not to place a decimal point at the beginning of a number, *e.g.*, the form .5 should always be replaced by 0.5. Tables should be prepared on separate sheets, numbered in the order in which they are to be used in the text, and instructions to the printer should be placed in the text to indicate where the tables are eventually to appear. In referring in the text to tables the number should be quoted and such phrases as "the following table" avoided. The number of illustrations should be kept as low as possible. In photographs much essential detail is lost and the inclusion of non-essential detail leads to confusion. Line drawings should be employed in preference to wash drawings or photographs as the cost of line blocks is about half of that of half-tones. Half-tone blocks have also to be printed on special paper which involves further expense. Illustrations should never be inserted in the MS., since they have to be sent to the engravers when the MS. is sent to the printers. They should be numbered consecutively. The descriptions of the figures should be written on separate sheets, but may also be inserted on the illustrations themselves. If drawings are made for reduction, the reference letters may be too small when reduced. It is usually better for the author to insert the lettering lightly in pencil so as to allow the skilled draughtsman employed by the engraver to give it its final form. The notes end by a consideration of the scientific names of animals and plants.

Ophthalmological Society of The United Kingdom.

The Ninth Annual Congress of the Society was held at the Royal Society of Medicine, 1, Wimpole Street, on Thursday, Friday, and Saturday, May 5, 6, and 7, 1921. The President, Mr. J. Herbert Fisher, was in the Chair, and the attendance was excellent. In spite of the difficulty of travelling in these days of coal shortage, many of the Provincial and Scottish Members were present, while Ireland was well represented by Dr. J. B. Story, the late President.

In opening the proceedings the President chose as his theme, the personal equation in clinical ophthalmology; he made a sympathetic allusion to the recent deaths of two of the original members of the Society, Mr. Higgens and Mr. H. E. Juler.

Dr. Maitland Ramsay read the first paper on "A case of Irido-cyclitis associated with swelling of the Parotid Glands." These cases are rare, or perhaps it would be better to say, that very few have been recorded. The Society was fortunate in having recorded in its *Transactions* an account of a similar case by Dr. George Mackay, P.R.C.S.Edin., in 1917. Dr. Mackay was unavoidably detained in Edinburgh by causes connected with an important surgical meeting there, but he sent by Dr. Traquair some further notes of his case. The case was discussed by the President, Drs. Hawthorne, Gray Clegg, and Percival. There was a consensus of opinion that such cases must be of toxic origin; in Dr. Maitland Ramsay's case a most searching examination had failed to detect the cause. Thyroid treatment was exhibited with very gratifying results.

Drs. Gray Clegg and Gordon Renwick read the notes of a case of partial monocular lamellar cataract associated with persistent pupillary membrane and injury at birth, and showed illustrations of the case upon the screen. The President, Sir William Lister, and Messrs. Collins, Mayou, Inman, and Goulden spoke on the subject.

Major Alexander read notes on the influence of age on the power of the crystalline lens, and a paper on the determination of cyclophoria at a near distance; difficult subjects, and the President and Dr. Percival were the only members present who were bold enough to contribute to the discussion.

Mr. R. R. James read a communication of an historical nature dealing with the life and works of Sir William Read, the noted quack oculist to Queen Anne.

Two papers sent by Dr. Wood, of Cape Town, on cases of flat neoplasm of the choroid, and on a case of symmetrical encroachment of the conjunctivae on the corneal margins terminated the morning business.

After lunch, a discussion was held on the psychology of vision in health and disease. Professor C. Spearman, in opening from the physical standpoint, gave a lucid address on the retinal corresponding

points. He spoke on the empirical and nativist theories of visual space, the "specific energy" of the nerve fibres, the theorems of Wheatstone, and other points, and brought forward some new experimental evidence on the subject of stereopsis, with a demonstration.

Mr. J. Herbert Parsons, F.R.S., was the opener from the ophthalmological side. He regretted that Sir Frederick Mott had been unable to open the discussion. His paper, which was a long one, dealt with the evolution of visual perceptions with special reference to the role of suppression.

Sir Robert Armstrong-Jones spoke on the comparative rarity of blindness in lunatics, in contradistinction to deafness; he detailed the results of experiments made at Claybury, many years ago, in which he had investigated the reaction of lunatics to coloured environment.

Dr. Kinnier Wilson dealt with the psychological peculiarities in some visual auras in cases of epilepsy. Mr. Frank Juler spoke on amblyopia from disuse: he brought forward a series of twenty-two cases of traumatic cataract occurring in childhood. His paper showed that, speaking generally, the earlier in life the accident occurred, the worse the visual result. After the age of six, given a healthy fundus and vitreous, and with no gross corneal scar in the line of vision, he found that a visual result of six-twelths and sometimes six-ninths could reasonably be expected. Dr. Percival also read a communication, while Dr. Harford entertained the company with some interesting diagrams of the psyche and soma, in cases of presbyopia and miners' nystagmus.

At 5 p.m. Mr. E. Treacher Collins delivered the Bowman Lecture in memory of the Founder. He took as his subject, "Changes in the Visual Organs, correlated with the adoption of arboreal life and the assumption of the Erect Posture," and held the attention of a very large audience for an hour and fifteen minutes.

The dinner of the Society was held at the Langham Hotel in the evening.

After an excellent dinner the President proposed the health of the Society: he asked those present to drink their own healths, which they did with much gusto; he said how proud he was to follow in the footsteps of his distinguished forerunners at St. Thomas's Hospital, Mr. Nettleship and Mr. Lawford. Mr. Richardson Cross gave the health of the Bowman Lecturer, spoke of Mr. Collins's tireless energy in whatever is for the good of the Society and his long connection with it, both in and out of office. Mr. Collins made a humorous reply, obstetrical in type.

Sir Anderson Critchett proposed the health of the President in a most felicitous speech and drawing a parallel between the doubting Apostle St. Thomas and Mr. Fisher, drew from the latter, in his

response, the statement that it is a question whether St. Thomas's Hospital may not have, in its name, an allusion to the martyred Archbishop of Canterbury.

On Friday morning a discussion was held on the treatment of manifest concomitant squint, opened by Messrs. Worth and Ballantyne. This is a subject of perennial interest; varied are the views as to the value of fusion training, the age at which operation should be recommended and the type of operation to be preferred. It provoked, as was to be expected, a good discussion, and Mr. Bishop Harman gave an excellent description illustrated by lantern slides of his reefing operation.

The clinical meeting was held at St. Thomas's Hospital in the afternoon. Mr. Fisher had been at great pains to arrange a good meeting. Among many exhibits by members of the St. Thomas's staff, special mention may be made of Professor Shattock's slides showing malarial parasites in the intra-ocular vessels, and Professor Parsons' anatomical specimens. The cases shown were numerous and interesting, and the Hospital and Florence Nightingale Home were open for inspection, while the Matron had kindly provided tea. Colonel Herbert brought up a series of cases to illustrate his iris prolapse operation in cases of glaucoma, on which he was to read a paper in the evening, while Dr. Maitland Ramsay demonstrated Barraquer's suction apparatus for the extraction of cataract. As we walked over Westminster Bridge on our way home, the shadows of two great ophthalmological heroes of the past were present in our imagination, William Cheselden and John Ranby. We were a little uncertain whether they would have been able to find their way about the modern St. Thomas's Hospital or whether they were hovering in the purlieus of London Bridge station in search of their old haunts; but that they were stirring that afternoon, we were convinced.

Some very important papers were read at the evening session. Colonel Herbert and Dr. Harrison Butler dealt with glaucoma. Messrs. Goulden and Whiting read the notes and showed lantern slides of a case of implantation cyst of the sclera following a squint operation. Sir William Lister spoke on intra-ocular epithelial cysts and also read a paper on punctate deposits on the retina. In each case he showed some really beautiful pictures of the preparations, both macroscopic and microscopic; he is to be congratulated, especially on his second paper, which places our knowledge of a very rare condition on a scientific basis.

On Saturday morning the usual business meeting was held after the Report of the Council of British Ophthalmologists had been read. The officers elected for the ensuing year are as follows:—President, Mr. Fisher; Vice-Presidents, Messrs. Story, Parsons, Grimsdale, and Cyril Walker; Treasurer, Sir Arnold Lawson;

Council, Messrs. Mayou, Brailey, Goulden, R. R. James, Traquair, and Harrison Butler; Secretaries, Messrs. Juler and J. F. Cunningham.

Mr. P. G. Doyne read an able paper on scotometry. He dealt with enlargement of the blind spot and scotomata in cases of chronic glaucoma, high myopia and tobacco amblyopia. Numerous charts were shown and the author is to be congratulated on a laborious and very useful piece of work.

Sir Arnold Lawson and Mr. Neame read the notes of a case of orbital tumour; the pathogenesis of this tumour is not absolutely settled yet, some further staining experiments being necessary; but it seems very likely that the case is one of neuroma of a ciliary nerve, a very rare thing indeed.

Mr. Grimsdale read a paper on the centre of rotation of the eye from the physiologico-anatomical point of view.

Mr. Stack showed a motor trephine, the motive force reminding one of the gyroscopic tops of one's childhood, and Dr. Traquair showed for Dr. Mackay two other forms of motor trephine.

A paper by Mr. Ormond on his method of suction extraction of cataract terminated the proceedings.

During Thursday and Friday the optophone was on view in the Bowman Library.

COUNCIL OF BRITISH OPHTHALMOLOGISTS

Annual Report

THE Council of British Ophthalmologists has to report that at the first meeting of the Session 1920-1921, Mr. J. B. Lawford was elected President in place of Sir Anderson Critchett, who filled the office with distinction during the first two years of the Council's work.

Sir George Berry and Mr. E. Treacher Collins were elected Vice-Presidents, Mr. J. Herbert Fisher, Honorary Treasurer, and Mr. Leslie Paton, Honorary Secretary. The following members were elected on the Executive Committee:—Messrs. Fisher, Collins, Parsons and Mayou, with the President and Secretary ex officiis.

It will be remembered that in 1919 the Council issued a report dealing with the training and examination of ophthalmic surgeons. It is gratifying to note that the following special examinations in ophthalmology have recently been established:—

M.S. London (Ophthalmology).

M.S. Victoria University, Manchester (Ophthalmology).

D.O.M.S. Conjoint Board, R.C.P. (Lond.), and R.C.S. (Eng.)

Also that at the present time a Committee of the Royal College

of Surgeons of England is considering the question of creating a higher form of diploma in connection with the Fellowship of the College.

During this Session the Council issued a report on the need for institutional treatment of children with contagious eye disease, which was forwarded to the Ministry of Health. Subsequently, a deputation from the Council was received by Sir George Newman, K.C.B., acting for the Minister of Health. As a result of this action of the Council, beds are now available at the Metropolitan Asylums Board Ophthalmia School at Swanley, for the treatment of London County Council school children suffering from ophthalmia or other contagious diseases of the eye.

The Council has had under its consideration the question of sight-testing by opticians, and has prepared and approved a report on the subject. It is not proposed to publish this report at present.

The Council has prepared a report on the notation of cylinder axes, and still has under consideration the question of the standardization of test types.

A member of the Council has been appointed to serve on a Joint Committee consisting of representatives from the Illuminating Engineering Society, the Research Association of British and Allied Manufacturers, the British Scientific Instrument Research Association, the Institution of Automobile Engineers, the Commercial Motor Users Association, and the Electric Lamp Manufacturers Association of Great Britain, Limited, on "Glare from Motor Car Head-lights."

The Council also agreed to appoint a Committee to consider the question of injuries to eye-sight resulting from the use of strong lights in cinema studios, but as the Minister of Health referred the matter to the Departmental Committee on the "Causes and Prevention of Blindness," the Council decided not to take any immediate action.

An interim report of the Committee on "Eyestrain in Cinemas," on which the Council has four representatives serving, has been issued. (See BRITISH JOURNAL OF OPHTHALMOLOGY, Vol. IV, p. 482, 1920.)

A Committee has been appointed to consider the Report of the Consultative Council of the Ministry of Health, in so far as it is likely to affect Ophthalmic Surgeons. This Committee has already gathered a considerable amount of information as to the work done by Ophthalmic Surgeons for various Public and State Authorities.

Another Committee is considering the question of examination, treatment and report of cases referred by Public Authorities to Ophthalmic Surgeons in Voluntary Hospitals.

As in previous years, the expenses of the Council have been defrayed by its members.

The Council has to thank the Council of the Royal Society of Medicine for affording them the use of a room for their meetings.

Report on the Institutional Treatment of London County Council school children suffering from Contagious Eye Diseases

The Council of British Ophthalmologists are glad to report that arrangements have been made for the treatment of children attending London County Council Schools who are suffering from contagious eye diseases.

In London there is a large number of cases of contagious eye diseases in children for which effective treatment is not provided. These include cases of trachoma, many cases of other forms of chronic ophthalmia, some accompanied by corneal ulceration, and a large number of cases of combined conjunctivitis and blepharitis. The cases are found mostly in the children of the poorest and least well-kept families of the community. They are well known to the school authorities, through whose visitors and Care Commitees they are sent to the various school clinics and out-patient departments of the hospitals. Notwithstanding the most careful and regular treatment at these centres, the cases drag out a long and miserable existence, mainly owing to the absence of proper treatment at home; even a daily attendance at a centre, or the services of a visiting district nurse, is often insufficient to effect a cure. A drastic change in the whole condition and environment of the children is needed, such as can be obtained by in-patient hospital treatment where good food and wholesome surroundings rapidly effect amelioration of these diseases. The pressure on children's beds in London hospitals is so great that only those children who show immediate danger of loss of sight, e.g., by perforation of the cornea, can be admitted.

The suffering which these conditions entail is often very serious; there is almost continuous pain, a state which is reflected in the whole aspect of the children.

The risk of communication of these diseases to others is very great, particularly in the poorest and most crowded households where they flourish.

The loss to the community by reason of the neglect of these cases is heavy. Of the children who are admitted to the London County Council "Myope classes or classes for Partly Blind Children," 30 per cent. are returned as being admitted on account of "damaged eyes from inflammation." In later years, these persons are of poor value in the economic life of the nation; not infrequently, they are a constant charge upon the community owing to their inability to do useful work.

Some years ago, the Poor Law Schools of London were hot-beds of contagious eye disease. Now these schools (notwithstanding

the massing of susceptible subjects) are relatively free from such disease. The change has been effected in the main by a system of isolation of infected or suspected cases. In 1903-4, the Metropolitan Asylums Board opened two schools for the isolation and institutional treatment of affected children. Accommodation was provided for about 600 children. Up to the year 1910, this accommodation was scarcely sufficient for the number requiring treatment. The efficacy of the régime of these hospital-schools has been proved by the steady decline in the number of cases requiring admittance since 1910, so that at the present time more than half the accommodation is unoccupied.

The experience of the benefits to be derived from prompt isolation and treatment of these eye affections is not confined to the M.A.B. schools, but is corroborated by similar experience in other countries.

(4) The hospitals of the M.A.B. have been in almost every instance provided at first for the treatment of the inmates of poor law institutions; gradually, there has been a widening of the scope of their operation, so that for fevers and certain other infectious diseases patients of other classes have been made eligible for admission.

Recently the M.A.B. has instituted a hospital for contagious eye diseases in the new-born, admission to which is not restricted to any one class of the community.

The Ophthalmia Schools of the M.A.B. at Swanley and Brentwood have only been used for those children whose parents are in receipt of poor law relief. As has been stated, there is now at these schools accommodation available for some hundreds of children. A return of the old defective conditions of the poor law schools is unlikely, so that these beds will not again be filled from that source.

There are many hundreds of children in the Metropolitan area to whom admission to these ophthalmia schools would be beneficial in the highest degree, and for whom there is no other suitable provision.

These arrangements have been made as a result of the report which the Council of British Ophthalmologists sent up to the Ministry of Health.

The institution at White Oak, Swanley, is under the Metropolitan Asylums Board and the number of children to be admitted will be dependent on the accommodation available.

Suitable cases should be referred to the district medical officer of the Public Health Department of the London County Council. The appropriate forms, which have to be filled up before admission, can be obtained from the School Medical Officer of the Public Health Department of the London County Council.

ABSTRACTS

I—VOLUNTARY CONTROL OF FUSION FACULTY

Barrett, Sir James (Melbourne).—A case of voluntary control of the fusion faculty. *Med. Jl. of Australia*, February 12, 1921, and *Med. Press and Circular*, April 6, 1921.

The interesting case reported by **Barrett** may well be given in the author's own words:

A.B., aged 28, was referred to me for ophthalmic examination. He was under observation for some mental disturbance with a suspicion of incipient general paralysis. The fundus and media were normal, and the following was the error of refraction:

Right eye ... ... cyl. + 0.5 90° = 6/4
Under mydriatic
Left eye ... ...cyl.+ 0.5 130° = 6/4

He had binocular vision. He possessed, however, the remarkable faculty of being able to dissociate the two eyes and to cause diplopia by moving either eye outwards, either separately or together. When he fixed with one eye and diverged the other, the deviation was about 20°.

He could not effect any vertical dissociation. He could fix with either the right eye or the left eye and move the non-fixing eye out 20°, or he could turn both eyes out at the same time each about 20°. He stated that he had been able to do this all his life, and that it was just the same thing to him as moving the hand. He possessed great powers of convergence and could converge to a point approximately five centimetres from the ocular base line. With the tropometer the excursion of the two eyes was as follows:

	Up.	Down.	Out.	In.
Right eye ...	25°	35 = 40°	50°	30°
Left eye ...	25°	35 = 40°	40°	40°

This is the only case that I have ever seen in which an individual had voluntary control over the eye movements, to the extent of being able to break the fusion faculty whenever he desired. There probably have been other instances, but I have been unable to lay my hand on an account of them. It is noteworthy that the fusion can only be broken by the action of the external rectus.

II—EXAMINATION OF ALBINO EYEBALL

Usher, C. H. (Aberdeen). — Histological examination of an adult human albino's eyeball, with a note on mesoblastic pigmentation in foetal eyes. *Biometrika*, Vol. XIII, No. 1, Oct., 1920.

When **Usher** took up the study of albinism in association with Karl Pearson and Nettleship some ten years ago, there was no recorded case of the microscopical examination of a human albino eyeball. Without such an examination it was impossible to say whether there was complete congenital absence of pigment or not, nor could the presence or absence of a fovea and of yellow colour at the macula be determined with any certainty. A further question of importance is that of the development of pigmentation in the foetus in connection with the theory propounded by Meckel and Mansfeld that the source of albinism is to be sought in an arrest of development. In the present paper Usher describes the eye of a human albino girl of 17 who died from meningitis complicated with epilepsy and mitral incompetence. Seven years previously she had attended the ophthalmic department, and, after the correction of a considerable amount of mixed astigmatism, had vision of 6/36 in each eye. A report of this case was published by Souter in "Albinism in Man," Pt. IV, No. 646.

Careful serial sections were cut horizontally, no sections being lost. In none of them could a fovea be made out, but the retina, as in normal eyes, had several layers of ganglion cells at the macula, The macula had a yellow colour when the eye was opened. The pigmentation of the retinal epithelium resembled that in a normal eye, but was less in quantity. Mesoblastic pigmentation was entirely absent in the iris, ciliary body, and most of the choroid, only a few cells in the macular region containing any pigment. Usher points out that the defective vision and nystagmus found in albinos is probably mainly due to the absence of a fovea, and not, as has been suggested, to defective retinal pigmentation. In the second part of the paper there is an account of the condition of the ocular pigmentation in some dark-raced foetuses. Usher sums up as follows:

"(*a*) Of the six albino eyeballs thus far examined, the albinos being determined clinically by the usual clinical characters, four are found to have pigment, of a fifth no statement was made by the examiner as to pigment. The sixth case was that of an *infant*, and here no trace of pigment was found in any of the structures of the eye. The remaining examinations of the human albino eye are concerned only with portions of the iris; in one no pigment, in the other some pigment was observed. The former case provides no evidence as to

whether the whole eyeball was or was not without pigment. We must conclude therefore that the total absence of pigment in the eye cannot be used as a definition of human albinism.

(*b*) As apart from absence of pigment, the absence or imperfect development of the *fovea centralis* shown to occur in albinotic eyes may possibly be the chief cause of defective vision in these cases.

(*c*) An examination has been made of the distribution of pigment in the eyes of a number of dark-raced individuals, adult, infant and foetal. The notes made indicate that in the dark-raced eye mesoblastic pigment appears earlier in foetal life, especially in the choroid, and is in much larger quantity by the time of birth than in the European eye.

According to this we should expect, if albinism consists of 'an arrest of development,' more pigment in albinos of dark races than in albinos of white races. Clinical examination of such albinos appears to indicate that this is a fact, but it is a fact which might be accounted for on other than the Meckel-Mansfeld hypothesis. At the same time it renders any definition of human albinism as a complete absence of ocular pigment less valid for any practical service in the study of the heredity of albinism."

Two coloured plates and an excellent bibliography accompany the paper.

Usher has made an important contribution to the study of both heredity and albinism, and one which all interested in either subject cannot afford to miss.

E. E. H.

III—SUPERNUMERARY TEETH IN ORBIT

de Lapersonne, Velter and Prélat (Paris)—Supernumerary teeth developing in the orbit. (Dents surnuméraires développées dans l'orbite). *Arch. d'Ophtal.*, March, 1921.

The case now recorded is believed by the authors to be unique; they have failed to find a similar example in their search through medical literature.

The patient, a well-grown healthy girl, aged 15, first came under observation in August, 1920, at the Hôtel Dieu, by reason of right-sided exophthalmos, and defect of vision dating from early childhood.

The right eye was slightly proptosed, and showed a high degree of deviation upwards and inwards, with complete loss of movement downwards and outwards. There was oscillatory nystagmus,

aggravated in attempts to look down. Refraction hypermetropic, vision with +2·D.Sph.=0·3. Media clear, fundus normal, pupillary reactions normal.

The left eye was in a normal position, its refraction and vision were similar to that of the right eye, and it also exhibited nystagmus.

The exophthalmos was not reducible nor pulsatile. There was slight facial asymmetry, the right superior maxilla being a little more prominent. No abnormality of the orbit could be detected by palpation. Rhinoscopy and diaphanoscopy failed to detect disease of the peri-orbital sinuses.

Radiography alone led to the diagnosis of the cause of the exophthalmos and the paralysis of ocular movements; the extraordinary conditions thus discovered are illustrated by two diagrams prepared from radiographs. One, in profile, shows the posterior part of the right upper dental arch with the molars developing normally; above this is seen a group of supernumerary teeth, in varying stages of development, some with one, some with two roots. They lie in different directions, vertical, horizontal, and oblique, and appear to be in the maxillary tuberosity. When they reach the floor of the orbit, they form an opaque mass in which it is difficult to distinguish the individual teeth.

Examination of the antero-posterior radiograph shows that the teeth in the orbit are larger and appear to be more highly developed. They occupy the deep part of the orbit, and some of the plates seem to show that they have traversed the posterior part of the spheno-maxillary fissure. This development explains the exophthalmos and the interference with the action of the inferior and external recti muscles.

The authors feel doubtful as to the exact number of supernumerary teeth in the maxilla and the orbit; their approximate estimate is twelve, but they recognise the possibility of a larger number of dental germs in the group, especially in the orbital portion and draw attention to the importance of this in relation to the ultimate development of the malformation.

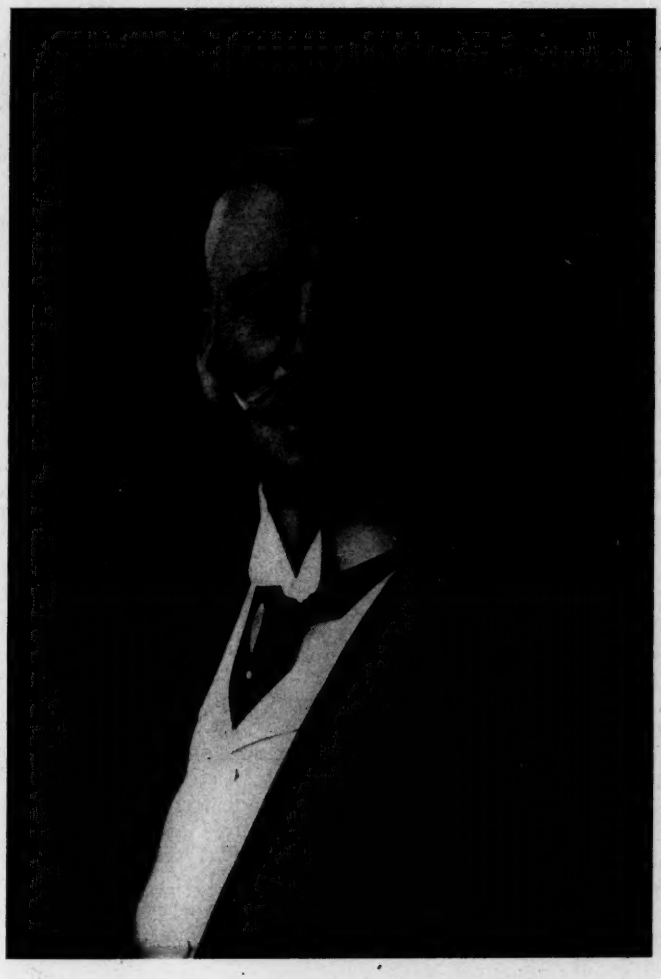
The question of treatment has been carefully considered. The teeth in the maxillary tuberosity are well tolerated, cause no inconvenience and do not call for interference. The orbital group, causing exophthalmos and displacement of the globe upwards presents a much more difficult problem. The decision reached is that surgical intervention ought to be postponed. The patient will be kept under observation in order that any extension of the orbital lesions, or any implication of the optic nerve may be detected.

J. B. LAWFORD.

OBITUARY

It is with deep regret that we announce the death upon April 23, at 17, Alexandra Court, London, of Henry Edward Juler, aged 79 years. Death took place from pneumonia, but Mr. Juler had been disabled by a crippling illness for some three years. The descendant of a Huguenot family, he received his medical education at St. Mary's Hospital, London, where he held the posts of demonstrator of anatomy (1877) and medical superintendent of the Hospital. While holding the former he had gained some experience of general practice by assisting a Dr. Gibson, at that time surgeon to Newgate Prison, and he then passed the examination for the fellowship of the Royal College of Surgeons of England. He studied in Paris and Berlin and decided to devote himself to eye work. Upon his return to London he was appointed clinical assistant to the Royal London Ophthalmic Hospital. He went into practice in Wimpole Street, and received the appointment of pathologist to the Royal Westminster Ophthalmic Hospital. In those days, although clinical material was abundant, there was no systematic teaching at that hospital, but Juler with the help of his colleagues speedily laid the foundation of a school of ophthalmology, and was the first to encourage students from Charing Cross Hospital to attend the neighbouring ophthalmic hospital. In 1884 he was appointed to the staff of St. Mary's Hospital as junior to Sir Anderson Critchett, and in 1901, when Sir Anderson retired, Juler became senior ophthalmic surgeon to the Hospital. Juler's chief contribution to the literature of ophthalmology was his "Handbook of Ophthalmic Science and Practice" (1884), a well-written, careful compendium, adorned with many illustrations. It was written when Juler was comparatively young, and firmly established his reputation as a keen clinical observer. It has gone through three editions in this country, to say nothing of an American edition. Juler was a frequent contributor to the *Transactions* of the Ophthalmological Society. His Harveian Lectures on "Syphilitic Diseases of the Eye and its Appendages" (1897) attracted a large circle of readers, and were based upon the wide experience he had gained as ophthalmic surgeon to the Lock Hospital in the Harrow Road. In 1879 Juler married Amy, the second daughter of Mr. W. J. Anderson, a merchant in business in Cape Town and London. Juler held office in several learned societies; the Harveian (president), the Ophthalmological (councillor and vice-president), British Medical Association, Royal Society of Medicine, Section of Ophthalmology (councillor) and the Oxford Ophthalmological Congress (councillor). Early in the war he lost his younger son,

George C. Juler, who was a lieutenant in the 5th Lancers. His surviving son Mr. Frank A. Juler, is assistant surgeon to the Royal London Ophthalmic Hospital and assistant ophthalmic surgeon to St. Mary's Hospital.



Mr. Juler is survived by his widow, a son, and three daughters, to whom we extend our heartfelt sympathy. Juler's outstanding personal qualities were geniality and generosity. He was never

happier than when entertaining his guests in Cavendish Square or elsewhere. He loved golf, his horse, and his motor car. A memorial service was held at All Saints, Margaret Street, and his mortal remains were cremated at Golder's Green.

The death occurred on February 15 last at Valetta, Malta, at the age of 75 years, of Surgeon-Lieut.-Colonel Manché, the first holder of the chair of ophthalmology in the University of Malta. He did much to develop ophthalmology in the island, where he established and maintained an ophthalmic clinic for poor patients.

NOTE

Committee on Lighting in Factories and Workshops

THE work of the Departmental Committee on the lighting of factories and workshops was given up after the issue of its first report owing to the conditions of war then prevailing. The work has been undertaken again, and the Committee hopes to arrive at definite standards of illumination needed in various kinds of work, and will extend the enquiry to industries not investigated before. The Committee, which has now been augmented, consists of the following members: Sir Richard Glazebrook, K.B.E., F.R.S. (Chairman); Mr. Leon Gaster (Hon. Secretary of the Illuminating Engineering Society); Prof. C. S. Sherrington, D.Sc., P.R.S. (Prof. of Physiology, University of Oxford); Mr. J. H. Parsons, D.Sc., F.R.S., F.R.C.S. (President of the Illuminating Engineering Society); Mr. W. C. D. Whetham, F.R.S. (Trinity College, Cambridge); Sir Arthur Whitelegge, K.C.B.; Dr. C. S. Myers, C.B.E., F.R.S. (Director of the Psychological Laboratory, Cambridge); Miss R. E. Squire, O.B.E. (H.M. Deputy-Principal Lady Inspector of Factories); Mr. D. R. Wilson, M.A. (Secretary of the Industrial Fatigue Research Board). The Secretaries to the Committee are:—Mr J. W. T. Walsh, M.A., M.Sc., of the National Physical Laboratory, Teddington, and Mr. H. C. Weston, M.J.Inst.E., Investigator to the Industrial Fatigue Research Board, to whom communications should be addressed at the offices of the Board, 6, John Street, Adelphi, W.C.2.